Society Proceedings

NEW YORK NEUROLOGICAL SOCIETY.

March 1, 1904.

The President, Dr. Pearce Bailey, in the Chair.

A Case of Facial Hemiatrophy.—This case was presented by Dr Pierce Clark. The speaker said he was indebted to Dr. McEntee for the history of the case, and to Dr. Holding for the X-ray pictures. The details of the case were as follows: The patient was a man, 62 years old, married, and the father of two children. His occupation was that of a clerk. His family history was negative. The patient himself had never had any serious illness. As a boy of five or six years he received a slight burn on the right side of the face, which, however, left no scar, and the occurrence of which slipped his memory during the first two or three examinations. Eighteen years ago, at the age of 44, the patient noticed a slight muscular contraction in the right side of the neck under the jaw, similar to that which was now present in the masseter and temporal muscles. The muscular twitchings were probably in the digastric and pterygoids. Rubbing diminished the spasm for a time. There was no pain, only discomfort, to which he soon accustomed himself. After three or four years he noticed the twitching, or fibrillations, now present. The tremor ceased at times, but quickly returned on fatigue, exposure to cold, excitement or any undue emotional stress. About six years ago he noticed the wasting in the right side of the face, Three years ago it became very marked, the teeth became decayed and loose, and were easily removed by a dentist. Early last January he contracted right trigeminal neuralgia in all three distributions of the nerve, from which he was now slowly recovering.

The physical examination showed that all the cranial nerves were free with the exception of the fifth. The sensory portion appeared intact, with the exception that differential smell and taste were a little slow on the right side, possibly due to the functional defect expressed in the neuralgia. The atrophy of the skin, subcutaneous fat and bone were sharply confined to the supposed distribution of the fifth nerve. There was no defect in the palate or tongue. There was no atrophy nor reaction of degeneration in the motor portion of the fifth nerve, the fibrillation being the only evidence of the involvement of the masseters and temporal. There was, however, a shortening of the masticatory muscle, which apparently restricted the separation of the jaws about one inch: this limitation was decreased in the absence of fibrillation so that he could do a little better than this, particularly in the morning, after a full night's rest. There were no lateral movements of the jaw, the pterygoids, apparently, not functionating.

The amount of asymmetry in this case was not so great as in those cases occurring in earlier life, where a hindrance of growth increased the effects of wasting. The atrophy had always been general and not especially marked in any one spot, as in the facial hemiatrophies of sclerodermal origin or of the morpheal type. The hair and the beard did not participate in the affection, although there was a rather marked thinning and whitening at the free border of the hair on the right side of the forehead. The lesion seemed to affect all divisions of the fifth nerve, and was not entirely confined to the right side.

Dr. William M. Leszynsky said he was interested to hear in Dr. Clark's case that the patient had developed a trigeminal neuralgia on the affected side of the face. The speaker said he had seen several cases of facial tic where subsequently a trigeminal neuralgia developed on the same side.

Dr. Pearce Bailey called attention to the long duration of the hemiatrophy, and said that the marked fibrillation was rather suggestive of the analogous condition observed in progressive muscular atrophy.

Dr. J. Ramsay Hunt said he did not see how Dr. Clark could exclude a slowly progressing lesion of the fifth nerve. A slowly growing tumor would account for the neuralgia, the hemiatrophy and fibrillation, and the muscular contractions in the masseter and temporal. The speaker said he thought the sensory symptoms excluded progressive muscular atrophy.

Dr Clark, in closing, said the analogy between his case and one of progressive muscular atrophy did not go further than the fibrillation. There was no evidence of progressive loss of power in the masseter and temporal muscles. If the lesion suggested by Dr. Hunt was a tumor it must be a very slowly progressing one, as the condition had existed for eighteen years. Possibly it had to do with the motor division of the fifth nerve only. Slowly progressive alterations in the posterior root ganglions were very likely to be attended with trophic loss without changes in the pain or tactile sensations.

Dr. Clark presented some X-ray photographs to illustrate an easy clinical method of demonstrating volumetric hypertrophy in bone, muscle and fat in parts which undergo true or false hypertrophy. It obviated the necessity of excising tissue, which was a painful and inaccurate means of determining these alterations, although the latter method must be resorted to in order to determine whether the muscular increase was due to hyperplasia or fiber hypertrophy. The photographs were of a case of hypertrophy of the leg of the paralyzed side of an infantile cerebral hemiplegia. The X-ray negatives were made by Dr. Holding.

A Case of Idiopathic Epilepsy in a Canary Bird.—This was reported by Dr. Pierce Clark. The bird was a male, one year old, from a brood of four. The family and personal history was negative. No cause for the epilepsy was known. Typical idiopathic *grand mal* of Jacksonian onset, and *petit mal* occurred irregularly every few days at the beginning of the observation period. A description of a *grand mal* attack was as follows: The bird gave an epileptic cry and fell backwards from the perch in a severe general tonic spasm, which lasted about thirty seconds; it lay very still on its left side where it had fallen. Then followed a very short clonic spasm, during which the head turned decidedly to the left side, with the legs fully extended. After the spasm the bird became violently automatic; it flew about the cage and dashed itself against the wires. After a few minutes of this sort of physical activity it lay quite still in a stage of coma for six or seven seconds, and then began to peep and stagger about the cage, still continuing its automatic, purposeless acts, such as treating the seed-cup as a water-cup, and *vice versa,* without partaking of the contents of either. After two minutes the attack gradually wore off, and the bird acted normally, although it appeared somewhat dazed and did not sing for the next few days. The following day, treatment was begun with a 1 per cent. solution of sodium bromide in the drinking water. Much improvement resulted in the next two weeks, the bird again beginning to sing and to act in a normal manner. Inasmuch as the amount of bromide solution was not limited, bromism began to appear in the third week. The bird sat with its head under its wing, with its feathers ruffled, and appeared sleepy and stupid. The bromide was then reduced to one drink a day at noon, which seemed quite sufficient. While the attacks during the next four months occurred weekly, they were all of a very slight character, excepting those of July 24, which were apparently induced by an accidental withdrawal of the bromides for a period of two weeks immediately preceding. These attacks, with the post-convulsive exhaustion paralysis, were as follows: The bird gave the epileptic cry and fell backwards from the perch, as before. The tonic spasm would last thirty seconds, with the legs drawn up so that the feet were under the wings. The bird lay on its left side, almost on the back; the head was turned to the left; the whole left side was strongly convulsed in the clonic phase, the legs extended, the wings spread to their fullest extent in fine tremor; then the spasm became remittent, and finally intermittent, that on the left side being the last to cease. The eyelids were in rapid nictation, the mandibles were open and shut rapidly in clonic spasm. The bird dashed about the cage as previously described, only more furiously; finally, it lay as though dead at the bottom of the cage (coma stage). The entire duration of the attack was four minutes.

Four more identical seizures followed the first in the next twenty minutes, a rather prolonged stupor stage ensuing after the last fit of the series. On attempting to walk and climb the perch the left leg could not be used. This temporary paralysis lasted about half an hour. The paralytic phenomenon occurred frequently after many subsequent attacks. It may be stated that when a bird extends the leg the toes are spread, but the converse was true in all the epileptic fits.

The bromides were finally withdrawn, and the attacks grew more severe and frequent, having a tendency to group themselves in series, a herald of the status epilepticus. The resulting paralysis in the leg sometimes lasted for an hour or more. The bird soon became exhausted by the frequent and severe attacks. He was no longer able to mount even the lowest perch, and sat on the bottom of the cage the greater part of the day with the head under the wing. The bromides were again given to note the possible staying power of the drug after the occurrence of the serial attacks. For a period of eight days the attacks ceased, and the bird gained steadily in strength and was apparently normal once more, when one day he was discovered in a peculiar position. He was tilted a little backward in the upright position, with the toes spread out for as broad a support as possible, and resting in part upon his tail. The head nodded rhythmically, and clonic spasms occurred every three seconds, accompanied by general fibrillary twitching of the entire body and rapid snapping of the mandibles with each clonic movement of the head. Rapid exhaustion ensued. During the last few hours the right leg and wing were so weak that he lay on the right side. The heart beat rapidly and violently at first, and finally weakened as death approached. There was apparently considerable increase in body heat, although thermometric measurement was not made. The autopsy, which was made ten minutes after death, showed an apparently normal brain in every respect, the calvarium also being normal in appearance. The brain was placed in absolute alcohol for cytological study of the cortex, but owing to some inexplicable error in the hardening process, the specimen failed to show anything convincing in its histo-pathological state.

The case showed (1) apparent idiopathic epilepsy in a bird. Nearly all previously reported cases of animal epilepsy, and especially in the bird, where an autopsy was made, proved to be Jacksonian traumatic epilepsies. (2) Post-epileptic exhaustion paralysis. (3) Status epilepticus and death analogous in many clinical respects to that seen in man.

A Case of Cerebral Tumor, with Specimen.—Dr. Clark reported this case in order to show the absence of focalizing symptoms, particularly astereogenosis, which one would expect to be present from the nature, size and position of the tumor. The history of the case was as follows: S. B., aged 36, a housewife, married two years. A native of Hungary. Her family history was negative, and she had always enjoyed good health until eighteen months ago, when she began to complain of post-occipital paroxysmal headache, which was especially marked on the left side and extended down into the corresponding shoulder. Six months later the attacks of headache were accompanied by vomiting. On August 13, 1903, one year after the onset of her symptoms, an examination was made, which showed that there was no cranial nerve involvement excepting the optic nerve, the power of which was impaired, and in November the patient became completely blind. There was no astereogenosis in hand or foot. There were no focalizing symptoms excepting the persistent pain over the left parietal region, which was increased on pressure and accompanied by stiffening of the muscles on the left side of the neck.

Inasmuch as the patient became delirious at times from the headache, and as the X-ray negative showed a light shadow approximately in the region of the pain and tenderness, it was decided to make an exploratory incision, which was done by Dr. Hartley at the New York Hospital on January 20, last. There was considerable bulging in the trephine opening, but the operation failed to disclose the tumor. However, it relieved the patient of headache and all her distressing symptoms. The swelling disappeared from the optic disc and the nerve showed complete atrophy. Three weeks after the operation the patient disturbed the dressing, which she said irritated her scalp. In consequence of this the wound became inflamed, and death resulted on February 24.

The autopsy was performed by Dr. Biggs, who found a tumor deeply imbedded in the posterior-superior part of the right parietal lobe. It was round and firm and attached to the neighboring portion of the falx. It was about four and one-half centimeters in diameter, anteriorily its border corresponded to the termination of the colloso-marginal fissure, and posteriorly it extended to within one centimeter of the parieto-occipital fissure. The probable diagnosis was an endothelioma of the dura.

A Case of Myasthenia Gravis Pseudo-Paralytica (?).—This was presented by Dr. B. Sachs. The patient was a man of 35; a picture framer by occupation. He had typhoid fever in childhood, and when he was 16 years old he had an attack of St. Vitus' dance, which lasted about four months and was very severe. Five years ago he had pneumonia. Between the ages of 16 and 30 he was, comparatively speaking, a well man. In April, 1902, he stated that his tongue felt very heavy; this lasted about six weeks and then passed off. On February 1, 1903, he received a glancing blow on the head with a sharp knife. The knife, which was about 40 inches long and weighed fully 75 pounds, was attached to a table above his head, and came down full force, although it only caused a slight wound, which readily healed within a week. In September, 1903, his tongue again began to feel heavy, and he became very nervous. At times he did not have the use of his tongue at all.

The first examination of the patient made by Dr. Sachs revealed a peculiarity of speech, which was thick and distinctly bulbar in character. The head was inclined forward, and to the right side. He also complained of weakness in his hands, and said he could not do his work properly. Formerly, he had been able to cut through many thicknesses of cardboard easily, but now he did not have the power to draw the knife through. There were no signs of paralysis, but he did not have the grasp of an able-bodied man. He complained of becoming tired after slight exertion. At the time of the first examination the knee-jerks were absent. Since December 5, 1903, when Dr. Sachs first saw him, there had been a distinct improvement in his speech and general condition. He complained that his vision was at times limited, so that he could not look up well. While using a knife or fork he occasionally halted in the movement for a moment, showing that the effort to use his muscles was at times ineffectual. Since the first examination the knee-jerks had returned and were normal. Another symptom of which he had complained was difficulty in swallowing; the food would at times produce a choking sensation and there was occasional regurgitation. He had also complained of drooling. He never had any trouble in chewing his food. There was no fibrillary tremor of the tongue; electrical examinations were negative. Heart and lungs and other organs were apparently normal. The reflexes were not increased. There was no atrophy anywhere, either in the shoulders or upper or lower extremities.

Dr. Sachs said he had examined this patient frequently, and the case had puzzled him greatly. The diagnosis had been still further obscured by the fact that the patient had been thoroughly mercurialized, and his gums had become affected. This was also the cause of the drooling. Among the conditions considered were a recurrence of the chorea, with thickness of speech, and amyotrophic lateral sclerosis, but the symptoms did not warrant either diagnosis. There was nothing to indicate the bulbar form of muscular atrophy, and Dr. Sachs said he finally came to the conclusion that the case represented an atypical form of myasthenia gravis pseudo-paralytica.

Dr. Bailey said that in cases of myasthenia gravis he had seen the muscles become easily fatigued. This applied not only to the voluntary muscles but to the reflexes.

Dr. Hunt said the symptoms in the early stage of the case, as the diplopia, paresthesia of the tongue and absent knee-jerks, were rather suggestive of the bulbar form of tabes. Dr. Spiller, of Philadelphia, had reported such a case of bulbar tabes about a year ago, in which there was difficulty in deglutition and articulation. The improvement in symptoms may have been due to the mercurialization; such retrogression in the earlier stage was not uncommon.

Dr. Sachs said that when he first saw the case he was inclined to regard it as an example of the bulbar form of tabes, but he had since been compelled to abandon that diagnosis. The knee-jerks, which were at first absent, had returned. There had never been any lightning pains nor eye symptoms nor other evidence of tabes.

Dr. I. Abrahamson called attention to the fact that the patient's speech was still of a jerky character, and that there was twitching of the mouth from time to time. This was suggestive of a recurrence of his chorea.

Dr. Bailey said he did not think it was possible to absolutely rule out multiple sclerosis.

Dr. Sachs, in reply to a question, said there had been no tremor nor nystagmus.

A Case of Multiple Neuritis.—This was presented by Dr. Isador Abrahamson. The patient was a woman, who was born in Germany thirty-four years ago. With the exception of an attack of pleurisy three years ago her personal history was negative. Her present illness dated back to February, 1903, when she noticed some weakness in the legs.

especially the right one. In March she had an attack of cough, with expectoration and pain, especially over the left shoulder-blade, and her attending physician told her she had a recurrence of her pleurisy. Three or four weeks later she had an attack of vomiting and diarrhea, with edema of the face and all four extremities; the latter symptoms were not marked; there was some fever. The urine was negative. A "leaky heart" was diagnosed by her physician. She now began to complain of pain, chiefly at night; it was boring in character, involving the outer sides of both legs. There were no local signs excepting tenderness on pressure along the outer surface of the legs. The pains and weakness in the legs progressed, and four weeks later she began to complain of numbness in the hands, especially the right, with beginning weakness on the thumb side more than on the ulnar. There was noticeable wasting in the legs, chiefly on their outer surfaces, and paresthesia in the region of the thighs, legs and feet. During all this time the patient remained in bed, suffering from frequent urination and diarrhea. Her fever lasted about two weeks. The diagnosis made at this time was influenza. Upon attempting to leave her bed she found that she was unable to walk. Dropped feet were apparent, and to a less extent dropped wrist. The physicians in attendance called the condition neuritis and partial muscular paralysis. According to their statement no sensory disturbances were present. The patient stated that she felt at all times the slightest touch, and that she was able to distinguish pin-pricks and hot and cold applications. She felt acutely the application of the faradic current. She was seen by Dr. Rieser on October 1, 1903, who found no sensory disturbances, although multiple neuritis of influenzal origin was diagnosed. The case was referred to Dr. Abrahamson on October 26, 1903. At that time some improvement had already taken place. There was marked motor weakness in both forearms and hands, and some wasting of the right thenar eminence. There was partial dropped wrist on both sides. The deep reflexes were absent; there was some ataxia, chiefly on the right side. The greatest weakness was in the extensors of the forearm, in the flexors of the hand and in the pronators and supinators. No sensory disturbances could be elicited. The heart was apparently normal and the trunk muscles intact. The abdominal reflexes were present.

There was distinct dropped foot, and the patient walked very unsteadily. There was some wasting of the peronei muscles. There were no fibrillary twitchings. The knee-jerks and Achilles reflex were absent on both sides. The feet were cold and somewhat congested. There was tenderness along the peroneal nerve; less along the posterior surface. Decided reaction of degeneration in peroneals. With the exception of slight tactile uncertainty, occurring only at times and not at all constant, there were no changes in tactile, pain or temperature sensations.

The patient was treated with electricity and strychnia and improved rapidly. She was now able to walk alone, although there was still some peroneal weakness.

In view of the history of edema, diarrhea and vomiting, the distinctly peripheral character of the paralysis, the paresthesia, pain, nerve tenderness, etc., the rather slow but progressive character of the onset left little doubt as to the diagnosis, in spite of the absence of objective sensory symptoms. Landry's paralysis, the speaker thought, could be excluded, though there were many points of resemblance. The etiology of the multiple neuritis, upon careful examination and questioning, negatived any metallic poisoning, and influenza had to be assumed as the etiological factor. A few similar cases had been reported by Blocq and others.

Dr. Sachs said if there was one fact that had been learned about multiple neuritis, it was that for some reason which it was difficult to explain sensory symptoms were almost invariably less prominent than the motor. He considered the motor form of multiple neuritis infinitely more common than the sensory. In the early stages of the disease there was usually some sensitiveness to pressure over the nerve, but actual objective sensory symptoms were frequently absent in the most pronounced and indubitable cases of multiple neuritis.

Dr. Leszynsky said he agreed entirely with Dr. Sachs. In several cases of multiple neuritis, however, he had observed slight sensory disturbances in the early stages, which disappeared within a few weeks. In the later stages of the disease he found no sensory symptoms whatever.

Dr. Clark said he had seen a number of Barnes' cases in London, and listened to a discussion upon them. They distinctly represented a form of polyneuritis, involving the motor fibers of the nerve almost exclusively. In the course of the discussion one of the speakers said he doubted whether there were any cases of purely sensory neuritis.

Dr. Abrahamson, in closing, said this was the only case of multiple neuritis he had seen that had not shown at any stage the least objective disturbance of sensibility.

A Case of Recurrent Peripheral Facial Paralysis.—This was presented by Dr. Abrahamson. The patient was a man, 32 years old, a hat-maker by occupation. His family history was negative. Six months ago a physician who examined him for a lodge found albumin in the urine. He denies syphilis. He used alcohol and tobacco in moderation. He stated that in the preparation of the felt for hats, mercury, and probably arsenic, was used for shrinking purposes, and that many of his fellow-workmen exhibited tremor and salivation. Personally, he had never shown any symptoms of mecurial poisoning.

The patient's symptoms were those of peripheral facial palsy, of which this was his fourth attack. His first attack was on the right side, and occurred sixteen years ago, all three branches of the nerve being involved. The attack lasted three or four months. His hearing remained normal; there was no otitis. The attack was attributed to exposure. He made a complete recovery; there were no sensory symptoms. His second attack occurred four years later. It was on the left side, and all three branches of the nerve were involved. It lasted about the same length of time as the first. There was no otitis; no sensory symptoms. Recovery was complete. The third attack was also on the left side, and occurred seven years ago. All three branches of the nerve were involved. There were no aural symptoms. The attack lasted four months; recovery was complete.

His present attack dated back to February 3, 1904. It began with pains radiating from the left aural region to the left lower face, and was followed by paralysis of all three branches of the left facial nerve. Hearing was not impaired; there were no evidences of any otitis. The patient also complained of some weakness and a cramp-like feeling in the left hand, especially when grasping objects. There was no headache, vertigo nor vomiting. His mentality was not impaired. There was no difficulty in swallowing, speaking, etc. Micturition and defecation were normal. He had never had diplopia.

Examination revealed a paralysis of all three branches of the facial nerve. The tongue was drawn slightly to the right; the uvula markedly so. Taste was impaired over the left half of the tongue. Hearing was not impaired. The ocular movements and superficial reflexes were normal. There was no ataxia. No evidence of lues. The sensibility, especially to temperature, was markedly diminished over the left half of the body, excluding the genitalia.

Dr. Abrahamson said the case was particularly interesting on account of the multiplicity of the attacks. As to the cause of the attacks, syphilis was apparently negatived by the absence of any history or findings indicating that disease, the vague nature of the ophthalmoscopic findings (which upon second examination were negative), the completeness of the facial palsy each time, the absence of involvement of any other cranial nerves, and the completeness of recovery without specific treatment. As to the cause of the attacks, mercury, with or without arsenic, was possibly the true etiological factor. The associated hemianesthesia was no doubt of a hysterical nature.

Nervous symptoms in Three Patients with Azoturia.—Dr. William H. Thomson read a paper on this subject, in which he stated that it was his routine practice, with all patients who consulted him for nervous ailments, to have frequent examinations made of the urine, for the purpose of determining the daily output of its *normal* ingredients. Repeatedly, in many cases of so-called neurasthenia, the reports would show that the excretion of urea would be only from one-fourth to one-half of what it ought to be, with sometimes a corresponding diminution of the other solids of the urine, and sometimes not, but rather an excess of the other saline ingredients with a marked diminution of urea.

Occasionally, however, he has had patients give a story with a series of symptoms resembling in many particulars those showing deficiency of elimination, but on examinatiion they proved to have a marked excess of both urea and solids, amounting in one case to ten times the quantity of urea of another patient who clinically might have been classed with him as a typical neurasthenic.

To illustrate this condition, Dr. Thompson briefly reported three such cases coming under his observation. The most interesting was that of a young man of 23, who was first seen on December 5, 1903. As he expressed it, he was suffering from a complete nervous breakdown, which came on him suddenly two years ago. He had an unfavorable neurotic family history; his father died of paralysis; his only brother was in an asylum, and one sister, six years older than himself, had attacks which, from his description, might be cyclical insanity. Another sister, 25 years old, was abnormally fat. The patient's story was that when he was 12 years old he had an attack of severe stomach trouble, in which he vomited constantly and became very much emaciated, and was told that his complexion became greenish. He was rapidly relieved of this by stopping all reading and by wearing glasses; he had been compelled to wear glasses ever since, and if by any chance he lost them, he began to feel sick at the stomach. During the past two years he had suffered from attacks of intense nervousness, with total inability to apply his mind to anything that required concentration of thought or attention. He had attacks also of gastric distress, but these were more intermittent. At other times he suffered from insomnia. He had no intestinal symptoms, the bowels moving regularly twice a day. His pulse was slow, weak and compressible. The temperature was normal. Examination showed nothing abnormal in the condition of his heart, lungs or other organs. The pupils were natural; no muscular tremors; the reflexes were normal.

Examination of his urine showed no abnormal ingredients of any kind, but the urea stood at the high figure of 51 grammes. As he was living on a very restricted diet, owing to his dyspeptic troubles, and as he could not take any hard muscular exercise, his output of urea should not have been above 20 grammes instead of 51. This urea excess was also accompanied by an output of total solids amounting to no less than

1,715.5 grains, or 114.37 grammes. Therefore, this patient, who had no fever whatever, no infection of any kind, and no diabetes mellitus, was, nevertheless, excreting more waste products daily than a case of severe typhoid fever. Under appropriate treatment the patient rapidly recovered, and he was now studying to pass a university examination.

In all three of Dr. Thomson's cases, pain in the eyes, especially from reading, was a leading symptom. What connection there could be between these ocular troubles and the marked excess in the excretion of urea and of other solids he could not say, and he was inclined to look upon their occurrence as accidental. His own view about the nature of these cases was that they were related to diabetes mellitus, and he would term them urea diabetes in distinction from saccharine diabetes. He did not venture to guess what the underlying cause of this particular form of systemic waste could be, but following his hypothesis he laid down for these patients the same line of treatment which he would for a diabetic. His notes showed that these patients could not digest starches, and for that reason he largely excluded farinaceous articles from their diet, and then prescribed the antiseptics, which he had treated of more fully in a recently published paper on the medical treatment of diabetes.

In reply to a question, Dr. Thomson said that in none of his three cases were the patients large meat eaters. The first patient said she could not eat meat with any comfort, and rarely touched it. The second patient said she had given up beef and mutton some years ago, because it usually gave her a headache. The third patient was almost an anchorite, and was practically starving himself, as he believed that his stomach was the cause of all his troubles.